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Paraquat (PQ) is a cationic nonselective bipyridyl herbicide widely used to control weeds and grasses in agriculture. Epidemiological studies have shown a strong correlation between paraquat exposure and Parkinson's disease (PD).
The orphan nuclear receptor Nurr1 is required for the development of the ventral mesencephalic dopaminergic neurons.
Paraquat, N-methyl-4-phenyl-1,2,3,6 tetrahydropyridine, and rotenone have been shown to reproduce several features of Parkinson's disease (PD).
Toxic concentrations of paraquat (0.2mM, 24h) caused death of both mature and immature cerebellar granule neurons (CGNs).
Paraquat (PQ) is suspected to be an environmental risk factor for Parkinson's disease (PD). A strong correlation between paraquat exposure and PD has been observed in several epidemiological studies.
Paraquat (PQ) causes selective degeneration of dopaminergic neurons in the substantia nigra pars compacta, reproducing a key feature of Parkinson's disease (PD).
An important feature of Parkinson's disease is the degeneration of dopaminergic neurons in the Substantia Nigra pars compacta (SNc).
Parkinson's disease (PD) is a neurodegenerative disease that mainly affects dopaminergic (DA-ergic) neurons in the substantia nigra.
BACKGROUND: Oxidative stress (OS) is an important factor in brain aging and neurodegenerative diseases. Certain neurodegenerative diseases, such as Parkinson's disease (PD), are characterized by increased OS.
Both epidemiological and pathological data suggest an inflammatory response including microglia activation and neuro-inflammation in PD.
Axonal degeneration is a common pathologic feature in peripheral neuropathy, neurodegenerative disease, and normal aging.
The effects of the 1-methyl-4-phenylpyridinium ion (MPP(+)) and some structurally related compounds on mitochondrial function and cell viability were investigated.
We have previously demonstrated that alpha-synuclein overexpression increases the membrane conductance of dopaminergic neurons.
Investigation of mechanisms responsible for dopaminergic neuron death is critical for understanding the pathogenesis of Parkinson's disease (PD).
Primary cultures of fetal rat cortical neurons and astrocytes were used to test the hypothesis that astrocyte-mediated toxicity contributes to dopaminergic neuron death.
This study determined how preconditioned neurons responded to oxygen-glucose deprivation (OGD) to result in neuroprotection.
The neurotoxins paraquat (PQ) and dopamine (DA or 6-OHDA) cause apoptosis of dopaminergic neurons in the substantia nigra.
Recent findings implicate the calcium-permeable transient receptor potential (TRP) melastatin subtype 2 (TRPM2) and calcium entry in the pathogenesis of neurodegenerative diseases.
The two hit hypothesis of neurodegeneration states that cells that have been severely stressed once are more vulnerable to subsequent insults.
Xenobiotic exposure is a risk factor in the etiology of neurodegenerative disease. It was recently hypothesized that restriction of xenobiotic exposure may be protective.
The herbicide paraquat is an environmental factor that may be involved in the etiology of Parkinson's disease (PD). Systemic exposure to paraquat causes neurodegeneration in animal models.

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Epidemiological and animal studies suggest that environmental toxins including paraquat (PQ) increase the risk of develop
The dual-hit hypothesis of neurodegeneration states that severe stress sensitizes vulnerable cells to subsequent challeng
Paraquat-stimulated NADPH-dependent lipid peroxidation in mouse brain and pulmonary microsomes was inhibited by s
Paraquat stimulates NADPH-Fe(2+)-dependent microsomal lipid peroxidation in mouse brain and strongly inhibits it in th
The cytotoxicity of reactive oxygen species and related agents toward cultured rat adrenal medullary pheochromocytom
We have investigated the response to oxidative stress in a model system obtained by stable transfection of the human n
In the central nervous system oxidative stress has been implicated in the pathology of several neurological disorders. The
The brain is particularly vulnerable to oxygen free radicals, which have been implicated in the pathology of several neuro
Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disorder characterized by the selective death of m
The last few decades have seen the marketing of hundreds of new pesticide products with a forecasted expansion of the
Oxidative stress has been considered as the leading cause of blood-brain barrier disruption which implicates many neuro
Filamentous inclusions of alpha-synuclein protein are hallmarks of neurodegenerative diseases collectively known as syn
The ability of brain tissue preparation to generate superoxide from xenobiotic interactions has been investigated. We sh
Nitric oxide synthase (NOS) oxidizes L-arginine to NO(&z.ccirf;) and L-citrulline. Recent studies have shown that this enzy
BACKGROUND: An increase in reactive oxygen species (ROS) burden and subsequent oxidative damage to nucleic acids, p
Carnosic acid (CA; C <sub>20</sub> H <sub>28</sub> O <sub>4</sub> ), which is also called salvin, is a major phenolic diterpene found in Rosmarinus officinalis L.
Pinocembrin (PB; 5,7-dihydroxyflavanone; C <sub>15</sub> H <sub>12</sub> O <sub>4</sub> ) is a flavonoid found in propolis and exerts antioxidant, anti-inflam
ETHNOPHARMACOLOGICAL RELEVANCE: Parkinson's disease (PD) is a multifactorial neurodegenerative disorder affectin

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Paraquat is a neurotoxic agent, and oxidative stress plays an important role in neuronal cell death after paraquat exposure.
Rat cultured cerebellar granule neurons (CGNs) were not sensitive to CuCl <sub>2</sub> (1-10 µM, 24 h), whereas paraquat (150 µM) induced cell death.
Mitochondrial oxidative stress is a contributing factor in the etiology of numerous neuronal disorders. However, the precise mechanisms of oxidative stress-induced neuronal death are not fully understood.
Cells from the midbrain micromass cell culture system from rat day 13 embryos were used to investigate the development of oxidative stress-induced neuronal death.
Objective To evaluate the protective effect of peanut sprout extract (PSE) against paraquat (PQ) induced SK-N-SH cells. Methods SK-N-SH cells were treated with PQ (100 µM) for 24 h. PSE (100 µg/ml) was added 1 h before PQ treatment. Cell viability was measured by MTT assay. ROS production was measured by DCF-DA fluorescence. Mitochondrial oxidative stress was measured by JC-1 fluorescence. Apoptosis was measured by Annexin V-FITC/PI flow cytometry. Results PSE treatment significantly protected SK-N-SH cells from PQ-induced cell death. PSE treatment significantly reduced PQ-induced ROS production, mitochondrial oxidative stress, and apoptosis. Conclusion PSE treatment protects SK-N-SH cells from PQ-induced cell death by reducing ROS production, mitochondrial oxidative stress, and apoptosis.
Purpose: To investigate whether two synthetic prenylated xanthone analogues -1,3,6,8-tetrahydroxy-9H-xanthen-9-one (1) and -1,3,6,8-tetrahydroxy-9H-xanthen-9-one (2) protect neurons from oxidative stress-induced cell death.
Oxidative stress caused by an increase in free radicals plays an important role in neuronal death. We investigated the effect of two synthetic prenylated xanthone analogues on oxidative stress-induced cell death in neurons.
Oxidative stress and apoptosis play pivotal roles in the pathogenesis of neurodegenerative diseases. We investigated the effect of two synthetic prenylated xanthone analogues on oxidative stress-induced cell death in neurons.
Background and purpose: Resveratrol (RSV) is a naturally existing polyphenolic compound abundantly found in grapes and other plants. RSV has been shown to have various biological activities, including antioxidant, anti-inflammatory, and neuroprotective effects.
Paraquat (PQ) through electron transfer reactions with NADH-dependent oxidoreductase of mitochondria and NADPH-dependent oxidoreductase of cytoplasm, produces superoxide anion (O <sub>2</sub> <sup>-</sup> ) and hydroxyl radical (OH <sup>•</sup> ), which are highly reactive and can cause oxidative damage to cellular components.
Amyloid $\beta$ -peptide (A $\beta$ ) and Paraquat PQ induce oxidative stress in astrocytes by formation of reactive oxygen species (ROS). We investigated the effect of A $\beta$ and PQ on oxidative stress in astrocytes.
BIOSIS COPYRIGHT: BIOL ABS. This study defined the ability of a large sample of heterogeneous pesticides and neurotoxins to induce oxidative stress in neurons.
1. Objectives Mutations in the PINK1 gene are responsible for autosomal recessive Parkinson's disease (PD). The project aims to investigate the role of PINK1 in PD pathogenesis.
Cytoplasmic inclusions known as Lewy bodies, a hallmark of Parkinson's disease (PD) pathology, may protect against cytoplasmic oxidative stress.
This study reports the effects of Ca <sup>2+</sup> channel blockers (Ca antagonists) on intraneuronal Ca <sup>2+</sup> ([Ca <sup>2+</sup> ] <sub>i</sub> ) movements and on the development of oxidative stress in neurons.
Parkinson's disease (PD) is a common neurodegenerative disorder and is characterized by the progressive loss of dopaminergic neurons in the substantia nigra.
BIOSIS COPYRIGHT: BIOL ABS. Cells from the midbrain micromass cell culture system from rat day 13 embryos were used to investigate the development of oxidative stress-induced neuronal death.

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